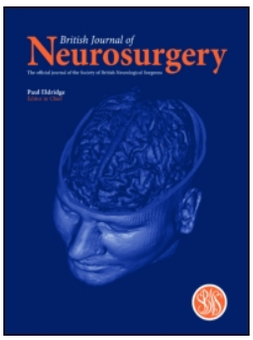

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RESEARCH ARTICLE

Resection extent of the supplementary motor area and post-operative neurological deficits in glioma surgery

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ABSTRACT

Objective The supplementary motor area (SMA) is important for the prediction of post-operative symptoms after surgical resection of gliomas. We investigated the relationships between clinical factors and the resection range of SMA gliomas, and the post-operative neurological symptoms. **Methods** We retrospectively studied 18 consecutive surgeries for gliomas involving the SMA proper performed in 13 patients. Seven cases were recurrence of the tumour. Clinical factors and details of specific resection of the SMA proper (resection of posterior part, medial wall) and cingulate motor area (CMA) were examined. **Results** Eight cases suffered new post-operative neurological deficits. Six of these eight cases had transient deficits. Permanent deficits persisted in two cases with partial weakness or paresis, after rapid improvement of post-operative global weakness or hemiplegia, respectively. The risk of post-operative neurological deficits was not associated with the resection of the posterior part of the SMA proper or the CMA, but was associated with resection of the medial wall of the SMA proper. Surgery for recurrent tumour was associated with post-operative neurological deficits. The medial wall was frequently resected in recurrent cases. **Discussion** The frequency of post-operative neurological symptoms, including SMA syndrome, may be higher after resection of the medial wall of the SMA proper compared with the resection of only the lateral surface of the SMA proper.

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Gliomas; recurrence; resection; supplementary motor area

Introduction

The supplementary motor area (SMA) occupies the medial portion of Brodmann cortical area 6 and is located in the superior frontal gyrus.[1–3] The anatomical boundaries of the SMA are defined by the leg region of the primary motor cortex posteriorly, the cingulate sulcus and genu of the corpus callosum inferiorly, and the edge of the medial cortex laterally. The anterior boundary is not well defined, but is located <5 cm from just before the leg region of the primary motor cortex.[3,4] The SMA represents a complex functional system for the initiation, execution and control of motor function and speech expression.[2] The SMA is important for the prediction of post-operative symptoms, including SMA syndrome after surgical resection of superior frontal gyrus lesions.[5] SMA syndrome may manifest as transient hemiparesis or motor apraxia with variable degrees of speech arrest followed by rapid recovery of neurological impairment.[4,6] However, the relationship between the extent of SMA resection and post-operative SMA syndrome has not been understood completely. The reported risks of post-operative neurological symptoms after resection of the SMA vary widely from 23% to 100%.[5,7–13] Opinions vary about the critical factors likely to risk the occurrence of post-operative SMA syndrome after surgical resection in this area. Post-operative SMA syndrome occurred in cases of 90% or more resection of the SMA.[11] The distance between the resected area and the precentral gyrus is significantly related to both transient and permanent neurological impairments.[7,10] Resection of the cingulate gyrus in addition to the SMA is significantly associated with the development of post-operative neurological impairment.[8]

On the other hand, various clinical risk factors may be associated with new post-operative neurological deficits after surgery for SMA glioma. Post-operative SMA syndrome occurred in a high proportion of patients after treatment for low grade SMA glioma.[11] However, second surgery for recurrent (high grade) glioma has been performed more often in recent years.[14,15] The largest increase in neurological risk occurs between the first and second surgery for gliomas in various regions.[15]

This study investigated the relationships between the extent of resection of a brain tumour involving the SMA and surrounding structures, and the post-operative neurological symptoms. The importance of various clinical factors, including tumour malignancy and recurrence, were also evaluated in surgery for SMA glioma.

Materials and methods

This study included 18 consecutive surgeries for glioma mainly located in the SMA performed in 13 patients between January 2007 and December 2013. Multiple surgeries in the same patient were treated as separate cases. In this series, Cases 1 and 13, Cases 2, 9 and 12, Cases 15 and 17, and Cases 8 and 16 occurred in the same patients. Data were retrospectively collected from clinical charts, surgical reports, operative electrophysiological and pathological reports and pre- and post-operative magnetic resonance (MR) imaging studies. These cases were evaluated to provide the information necessary to investigate the relationship between the resection area and post-operative neurological symptoms.

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The boundaries of the SMA were defined posteriorly by the precentral sulcus, inferiorly by the cingulate sulcus, laterally by the superior frontal sulcus and anteriorly by the most rostral point of the genu of the corpus callosum.[1–3] The anterior pre-SMA and posterior SMA proper were divided by the vertical commissure anterior (VCA) line.[8,16] In this study, cases of resection of only the pre-SMA were excluded and the SMA proper behind the VCA was defined as the true SMA for several reasons discussed later.

This analysis was limited to only patients with no pre-operative motor deficits or mild motor weakness. The 10 males and eight females were aged from 15 to 66 years at the time of operation (mean age 39.5 years). All patients were right-handed. Pre-operatively, 13 patients had no motor deficits and five patients had mild contralateral motor deficits. Four patients suffered from seizure. Seven patients underwent surgery for recurrence, including four patients who had undergone radiotherapy previously. The histological diagnosis was glioblastoma in seven patients, oligoastrocytoma in five, anaplastic oligoastrocytoma in three, diffuse astrocytoma in two and anaplastic oligodendroglioma in one. All clinical data collected for this study are shown in Table 1. This study was reviewed and approved by the institutional review board of Gunma University Graduate School of Medicine.

Surgical procedure and cortical electrical stimulation

All patients underwent open surgical resection under general anaesthesia with continuous infusion of propofol for intra-operative motor and sensory monitoring. No muscle relaxant was used except during induction of anaesthesia. After craniotomy and dural opening, a four-channel strip electrode was positioned subdurally on the cortex of the rolandic region. After positioning of the strip electrode, the median nerve was stimulated and the central sulcus was identified by N20 somatosensory-evoked potential phase reversal.[17] Monitoring of the motor evoked potential (MEP) was performed intermittently using the strip electrode by anodal direct cortical stimulation of the precentral gyrus until the end of resection.[18] The compound muscle action potentials were detected with disposable subdermal needle electrodes, which were placed in the standard muscles of the contralateral side to the tumour as follows: the biceps, forearm flexors, thenar, oris and anterior tibialis muscles.[19,20] The MEP was also monitored using a monopolar electrode as the resection approached any primary motor area and the resection cavity (pyramidal tract). Primary motor area stimulation was performed by applying anodic five-train rectangular pulses, with stimulation width of 0.2 ms, intensity of 4–20 mA, and frequency of 1 Hz.[19] Direct subcortical stimulation was applied to the wall of the resection cavity under the same conditions with intensity of 0.5–25 mA.[20] The procedures of the tumour resection and closure followed the established standards for care. At the completion of resection, MEPs had recovered to above 50% of the value before resection in all patients.

Radiological evaluation

The extents of both the tumour and the resection area were carefully evaluated on post-operative MR images, with reference to the pre-operative MR images. Post-operative MR imaging with or without contrast medium was performed within a month of surgery to evaluate the resected area. The central and precentral sulci were identified on the MR images by examining the typical anatomical landmarks.[21–23] To confirm the eloquent cortical areas, 15 patients underwent pre-operative functional MR imaging to detect the central sulcus and the hand-knob area using the finger tapping task.[23] The anatomical limits of the SMA were defined as the precentral sulcus posteriorly, the cingulate sulcus and genu of corpus callosum inferiorly and superior frontal sulcus laterally.[1–3] The SMA proper was defined as the true part of the SMA posterior to the line vertical to the anterior commissure–posterior commissure plane and crossing the anterior commissure, or the VCA line (Figure 1a).[8,16] The SMA proper was also divided into the lateral surface (horizontal portion) and the medial wall (vertical portion) in orthogonal directions (Figure 1b).[24–26] Furthermore, the SMA proper was divided into the anterior (rostral) part, and the posterior (caudal) part in the longitudinal direction in this study (Figure 1c).[27] The cingulate motor cortex (CMA) spans both banks of the cingulate sulcus, which are mainly located at the same rostro-caudal

Table 1. Clinical, radiographic and surgical features in 18 procedures.

						Resection			
No.	Age/Sex	Side	Previous treatment	Presentation	Diagnosis	Posterior parts of the SMA proper	Medial wall of the SMA proper	CMA	Immediate postoperative neurological deficits
1	23/M	Left	–	–	OA	–	–	–	–
2	32/F	Left	Resection + RT	–	GBM	+	–	–	–
3	32/M	Right	–	–	OA	+	–	–	–
4	52/M	Right	–	–	AOA	–	–	+	–
5	53/M	Right	–	Motor weakness (4/5)	GBM	–	–	+	–
6	61/F	Right	–	Motor weakness (4/5)	GBM	–	–	+	–
7	45/F	Right	–	Motor weakness (4/5)	AOD	+	–	+	–
8	64/F	Left	–	Hemiparesis (3/5)	GBM	+	–	+	–
9	30/F	Left	–	Seizure	AOA	–	+	+	–
10	27/M	Right	–	Seizure	DA	–	+	+	–
11	22/F	Right	Resection	Seizure	OA	+	+	–	Fine motion disorder[a]
12	32/F	Left	Resection + RT	–	GBM	+	+	+	Hemiparesis (2/5), speech hesitancy[a]
13	27/M	Left	Resection	–	OA	–	+	+	Hemiparesis (2/5), speech arrest[a]
14	62/M	Left	–	–	GBM	–	–	+	Hemiplegia (1/5), speech arrest[a]
15	15/M	Left	–	Seizure	DA	+	+	–	Hemiplegia (1/5), speech arrest[a]
16	66/F	Left	Resection + RT	Hemiparesis (3/5)	GBM	+	+	+	Hemiplegia (1/5)[a]
17	17/M	Left	Resection	–	OA	+	+	+	Motor weakness (4/5)[b]
18	51/M	Left	Resection + RT	–	AOA	+	+	+	Hemiplegia (1/5)[b]

AOA = anaplastic oligoastrocytoma; AOD = anaplastic oligodendroglioma; CMA = cingulate motor area; DA = diffuse astrocytoma; F = female; GBM = glioblastoma multiforme; M = male; OA = oligoastrocytoma; RT = radiation therapy; SMA = supplementary motor area.
[a]Patients experienced post-operative transient neurological deficits.
[b]Patients experienced partial recovery and residual permanent deficits.

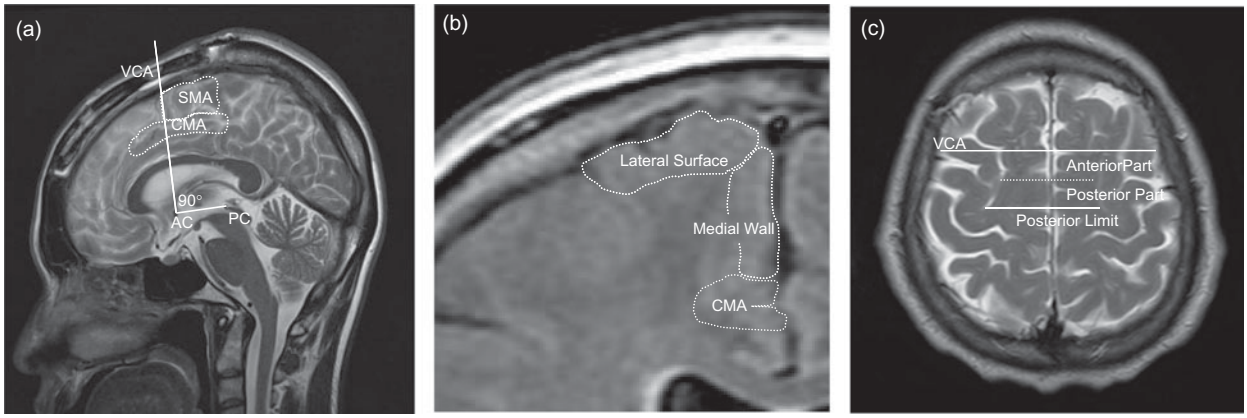

Figure 1. Procedure for evaluation of supplementary motor area (SMA) resection. (a) The anterior commissure (AC) and the posterior commissure (PC) are identified on the midsagittal MR image. The lines are perpendicular to the AC–PC line through the AC, or the vertical commissure anterior (VCA) line. The SMA proper was defined as the part caudal to the VCA line. The cingulate motor cortex (CMA) spans both banks of the cingulate sulcus, mainly located at the same rostrocaudal level as the pre-SMA and the SMA proper. (b) The SMA is shown on an enlarged coronal MR image. Lateral surface, medial wall and CMA are shown in enclosed areas with dotted lines. (c) The inverted omega is identified on the axial MR image. The SMA proper is divided into anterior and posterior parts longitudinally.

level as the pre-SMA and the SMA proper (Figure 1a and b). These areas were surgically removed as confirmed on post-operative axial, sagittal and coronal MR images. The extent of the resection was evaluated as including the posterior part of SMA proper, the medial wall of the SMA proper and the CMA on axial, coronal and sagittal MR images, and traced on the standard brain model.[5]

Post-operative findings and evaluations

The neurological status of each patient was assessed pre-operatively and post-operatively at 1-d intervals until the patient was discharged. The follow-up period after surgery was six months. Post-operative motor and language deficits were evaluated by the neurosurgeons responsible for each surgery, including the authors (Y.I., K.H. and K.S.), with reference to the assessments of the responsible physician specialising in physical medicine. Motor deficits were rated using the WHO motor grading scale: 5/5, normal power; 4/5, active movement against gravity and resistance; 3/5, active movement against gravity; 2/5, active movement only in the absence of gravity; 1/5, trace contraction or 0/5, no contraction.[28] In this study, hemiplegia was defined as 0–1/5, severe hemiparesis as 2/5, mild hemiparesis as 3/5 and motor weakness as 4/5. Complete SMA syndrome was defined as contralateral hemiplegia with or without mutism, and partial SMA syndrome was classified as contralateral hemiparesis and/or speech hesitancy.[11] Additional post-operative deficits that did not improve during the six-month follow-up period were recorded as permanent neurological deficits.[11]

Several clinical factors, including patient age, previous resection, pre-operative motor deficits, tumour grade, resected area and resection of more detailed parts of the SMA (posterior part and medial wall of the SMA proper) or CMA as a SMA-related region in the medial wall, were evaluated as risk factors for the development of post-operative neurological deficits following SMA glioma resection.

Statistical analysis

The relationships between new post-operative neurological deficits and each risk factor were examined by Fisher's exact probability test. A value of $p < 0.05$ was considered to be statistically significant.

Results

The clinical, radiographic and surgical features are summarised in Table 1. Eight patients presented with post-operative neurological deficits. Six of these eight patients had transient deficits (three with complete SMA syndrome and three with partial SMA syndrome), and two had permanent deficits. Just after surgery (<1 d), four patients experienced hemiplegia on the contralateral side, two patients experienced hemiparesis, predominantly affecting the upper extremities, one patient experienced motor weakness of the lower extremities and one patient experienced disorder of fine motion in the contralateral hand. Four of the 11 patients who underwent resection in the dominant hemisphere experienced speech deficits (speech arrest in three and speech initiation difficulties in one). Several weeks later, these deficits had improved significantly in five patients. Case 11 with disorder of fine motion showed full recovery in one week. Two of the four patients with hemiplegia fully recovered (Cases 14 and 15). The two patients with hemiparesis both had good recovery. Case 16 with hemiplegia regained some finger voluntary movement in two weeks. However, her hemiparesis of the proximal extremities was prolonged. She underwent rehabilitation for three months, after which her hemiparesis had improved to the same level before the operation. Two of the eight patients had permanent deficits. Case 17 suffered permanent ankle weakness with limping gait. Case 18 with hemiplegia had partial recovery and experienced residual hemiparesis of the right shoulder and arm. All patients with speech deficits recovered in one month.

Post-operative MR imaging findings of all patients are shown in Figure 2. The extent of the resection using tracing on the standard brain model is shown in Figure 3. Resection of the medial wall of SMA proper was clearly indicated in cases with post-operative neurological deficits.

The relationships between post-operative neurological deficits and clinical characteristics are shown in Table 2. New post-operative neurological deficits occurred in cases with specific risk factors as follows: high age (>50 years) 3/7 and low age (<50 years) 5/11, past history of resection 6/7 and no history 2/11, pre-operative motor weakness 1/5 and no motor deficits 7/13, WHO grade II (low grade) 4/7 and WHO grade III or IV (high grade) 4/11, resection of the posterior part of SMA proper 6/10 and resection of only the anterior part 2/8, resection of the medial wall of SMA proper 7/9 and resection of only the lateral surface of SMA 1/9, resection of both posterior part and medial wall of SMA proper 7/9 and others 1/

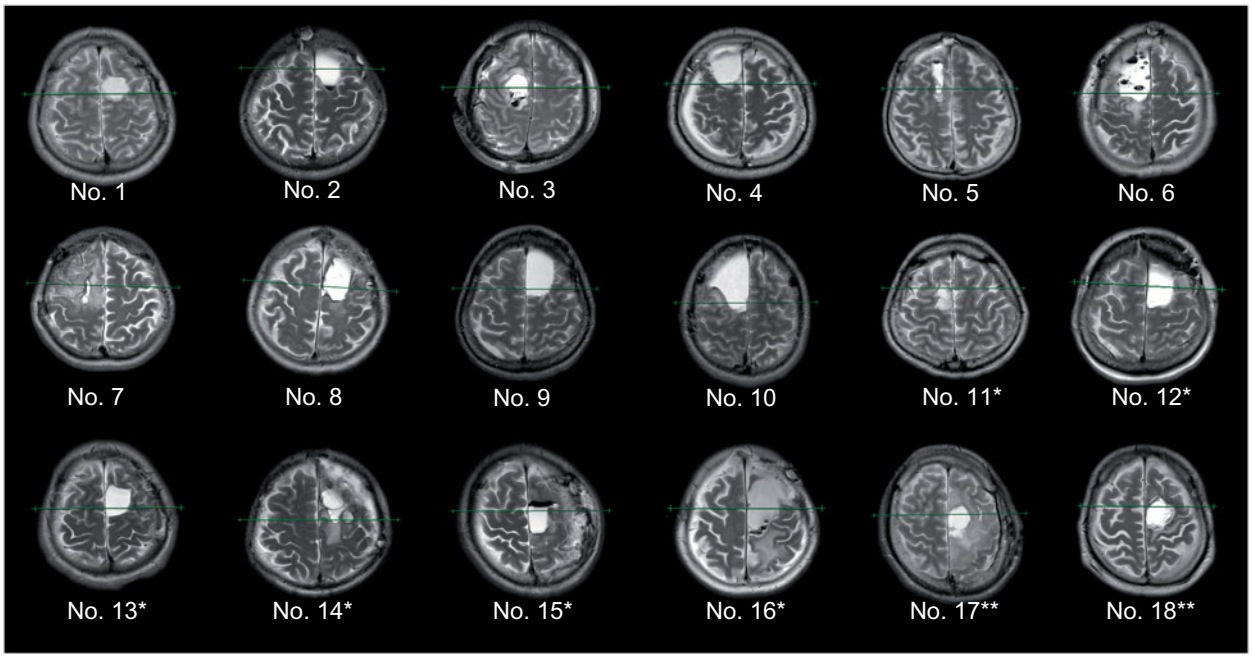

Figure 2. Post-operative axial T2-weighted images obtained in all patients showing the landmark of the central sulcus, the inverted omega. Single asterisk represents patient who experienced post-operative transient neurological deficits. Double asterisks represent patient who experienced post-operative permanent neurological deficits. VCA = vertical commissure anterior.

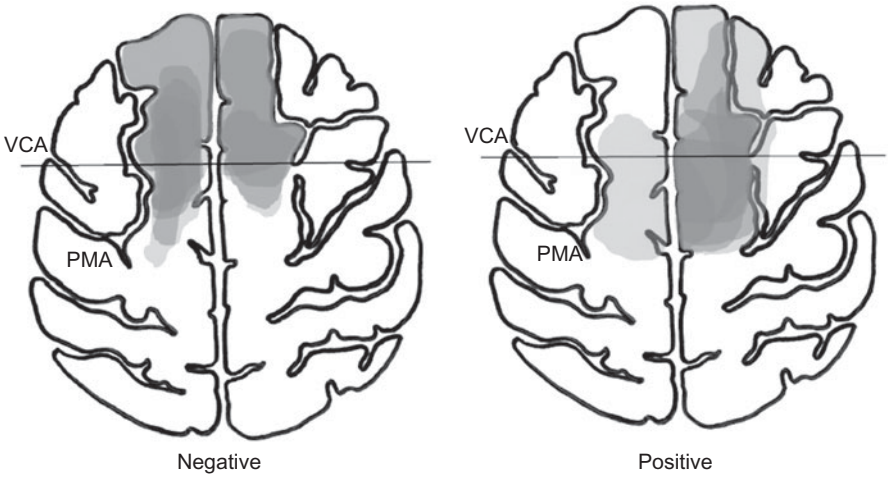

Figure 3. Schematic representation of tumour resections. The extent of tumour resection is indicated as the shaded area, and transferred to a schematic axial brain section. Negative (Left): diagram obtained in patients without post-operative neurological deficits. Positive (Right): diagram obtained in patients with post-operative deficits. PMA = primary motor area;VCA = vertical commissure anterior.

9, and resection of CMA 6/13 and intact CMA 2/5 (Table 2). Statistical analysis revealed that past history of resection ($p = 0.01$), resection of the medial wall of SMA proper ($p = 0.02$), and resection of both posterior part and medial wall of SMA proper ($p = 0.002$) were significantly associated with post-operative neurological deficits.

Extensive resection of SMA-related regions in the initial resection or resection for recurrence were examined. Extensive resection of SMA-related regions was performed in the initial resection or resection for recurrence as follows: resection of the posterior part of the SMA proper 4/11 (initial) and 6/7 (recurrence), resection of the medial wall of the SMA proper 3/11 (initial) and 6/7 (recurrence) ($p < 0.05$), resection of both posterior part and medial wall of SMA proper 1/11 (initial) and 5/7 (recurrence) ($p < 0.05$) and resection of the CMA 8/11 (initial) and 5/7 (recurrence). The frequency of medial wall resection was higher in surgery for recurrence. The resection included both posterior parts and medial wall of the SMA proper in 6 of the 18 cases (33%). All these cases suffered post-operative neurological deficits. Five of these six cases (83.3%) underwent surgery for recurrence.

Discussion

SMA syndrome has been reported to evolve in three specific stages: global akinesia occurs immediately after surgery, more prominent contralaterally with speech arrest; sudden recovery occurs a few days later, with persistent reduction in contralateral motor activity,

Table 2. Relationships between post-operative neurological deficits and clinical characteristics.

	Post-operative motor deficits			
	Present	None	Total	*p* Value
Age				1.00
>50 years	3	4	7	
<50 years	5	6	11	
Previous treatment				0.01
Resection and/or RT	6	1	7	
none	2	9	11	
Pre-operative motor deficits				0.31
Yes	1	4	5	
No	7	6	13	
Tumour grade[a]				0.63
Low	4	3	7	
High	4	7	11	
Resection of the posterior parts of SMA proper				0.19
With	6	4	10	
Without	2	6	8	
Resection of the medial wall of SMA proper				0.02
With	7	2	9	
Without	1	8	9	
Resection of both posterior part and medial wall of the SMA proper				0.002
With	6	0	6	
Without	2	10	12	
Resection of the CMA				1.00
With	6	7	13	
Without	2	3	5	

CMA = cingulate motor area; RT = radiation therapy; SMA = supplementary motor area.
[a]Tumour grade: low = WHO grade II; high = WHO grade III or IV.

emotional facial palsy and reduction in spontaneous speech; and the only persistent sequela is disturbance of the alternating movements of the hands within weeks to months after surgery.[4] This spectrum of severe but transient deficits after compromise of the ipsilateral SMA has been termed SMA syndrome.[4,11] Transient and various lesser degrees of contralateral paresis and/or speech impairment compared with the severity of classical SMA syndrome may occur in patients after SMA resection, which is called partial SMA syndrome.[11] Permanent paresis could occur after SMA resection, possibly caused by injury to the motor cortex or pyramidal tract.[7,8,10,11]

In our series, permanent weakness or paresis persisted in two patients with recurrent gliomas, who had not presented with pre-operative severe paresis. One patient (Case 17) had weakness of the entire contralateral leg immediately after surgery, which improved after one week. However, weakness of the ankle joint persisted. Another patient (Case 18) had contralateral global akinesia including the upper and lower extremities immediately after surgery, which improved after one week. However, paresis of the shoulder joint persisted. The severe motor impairment, which had occurred immediately after surgery improved quickly in Case 18. We felt that the symptoms just after surgery in these cases might represent combined SMA-related transient paresis and permanent injury to the pyramidal tract. These patients had undergone partial or subtotal tumour resection including the SMA, including the posterior part and medial wall of the SMA proper without definite cortical resection of the precentral gyrus. Intra-operative MEP monitoring showed no changes in either patient. However, white matter injury of the primary motor fibres, such as the corticospinal tract other than the part covered by standard MEP monitoring, could have caused the partial but permanent neurological symptoms.[7,8,10] All these patients had past history of resection, with recurrence of the tumour near the wall of the past resection cavity, so tumour removal may have extended closer to the corticospinal tract and the primary motor area. Both posterior part and medial wall of SMA were resected in both cases. This possibility is discussed later.

The pre-SMA does not project in contrast with the SMA proper that projects strongly to the anatomical primary motor area. Conversely, the pre-SMA receives strong input from the prefrontal area, whereas the SMA does not receive any input.[29,30] Therefore, the pre-SMA is considered to be a different region from the SMA proper, and a higher control centre than the SMA proper for many reasons.[2,31] The anterior border of the pre-SMA has been variously defined.[8,11,31] In this study, the SMA was strictly defined as located posteriorly from the VCA line, and the series included only cases of surgical resection of the SMA proper.[2] Based on this definition, we found that some cases unexpectedly did not suffer SMA syndrome despite partial resection of the SMA proper. To clarify this finding, we examined the details of resection of the specific parts of the SMA proper. First, we classified the SMA proper into rostral and caudal parts in the longitudinal direction at the halfway line.[27,32] Second, each case was classified according to whether resection of the medial wall was performed. Furthermore, resection of the CMA, a region in the medial wall related to the SMA, was also examined.[8,33,34] We found that post-operative neurological symptoms including SMA syndrome easily occurred after resection of the medial wall of the SMA proper. Conversely, post-operative neurological symptoms were uncommon if the medial wall remained intact, even after resection of the posterior part of the SMA proper or CMA.

In our study, post-operative neurological symptoms appeared in many cases after resection of the SMA proper medial wall. Such findings are considered to be acceptable and not surprising outcomes based on the results of previous research about the somatotopy of the SMA.[24] The SMA proper is a motor-related region on the frontal medial wall and extends partly onto the surface of the hemisphere laterally just anterior to the primary motor leg representation.[3,4] Forelimb representation in the SMA appears mainly on the medial surface of the hemisphere based on intracortical microstimulation mapping using rhesus monkeys.[25] The main functional area is located in the medial wall except for a limited part of the hindlimb extending into the caudal end of the lateral surface.[24,26] Several experimental studies have focused only on the medial wall.[35–37] However, the importance of discriminating the medial wall of the SMA proper from the lateral surface has never been reported in clinical neurosurgery.[5–13] Many neurosurgeons understand the SMA proper as a region in front of the primary motor cortex, including the lateral surface of the superior frontal gyrus. Neurosurgeons first observe the convexity brain surface, and then identify the central sulcus and motor area in the surgery of SMA lesions. Therefore, the medial wall of SMA may not receive sufficient attention because of the presence of the superior sagittal sinus, bridging veins and dural opening procedure. The medial wall of the SMA proper can be observed only after dissection of the interhemispheric fissure, and the direction of the medial wall is difficult to determine in the operative field. Resection of the medial wall cortex is one of the important decisions to be made during the final stage of tumour resection in this area. The resection area of the glioma may depend on various factors, including tumour grade, invasion area, pre-operative symptoms, monitoring results, prognosis and so on. The findings of this study may have important implications in surgical decision-making in the resection of SMA glial tumours.

The CMA is a motor area located on the upper and lower walls of the cingulate sulcus, and is distinguished from the cingulate

gyrus in the inner wall (Figure 1).[33,36] In recent years, anatomical connections between this area and the spinal cord, primary motor area and SMA have been proposed.[33,36] However the neurophysiological functions of the CMA are not well understood. Resection of the cingulate gyrus is a risk factor for post-operative neurological symptoms.[8,34] However, such post-operative neurological symptoms may have been caused by SMA resection in the surgical trajectory to remove the cingulate gyrus.[8,34] Therefore, a similar mechanism might occur in the resection of the CMA. Among our six cases of CMA damage with post-operative symptoms, five cases (5/6, 83%) had associated SMA medial wall resection. This finding may be consistent with previous reports about cingulate gyrus.[8,34] However, no definite conclusion can be drawn based on this small series of cases. In this study, CMA damage was examined only in cases with SMA proper damage. Experience with larger series is required.

In recent years, the necessity for second surgery has increased as a factor of multi-modality therapy because of the remarkable progress in effective chemotherapies, such as temozolomide and other molecular targeting agents.[14,15] However, the risk of post-operative neurological complications increased by 2.5 times at second resection for recurrent glioma compared with initial resection, and paresis was the most common neurological complication.[15] The relationship between post-operative neurological symptoms and discrimination of initial or recurrent tumour was not investigated in the previous series of SMA surgery.[10,11,13]

In our series, all patients with permanent neurological defects after the operation had all undergone surgery for recurrent tumour including malignant tumour. In contrast, no patient had post-surgical permanent neurological symptoms after initial surgery. Post-operative neurological symptoms were transient after initial surgery, and could be considered to represent SMA (related) syndrome. In contrast, this study found relatively high risk for post-operative neurological symptoms after surgery for recurrent glioma in the SMA even if advanced technology, such as MEP monitoring, is used. The reason may be simple. The most common regional recurrence pattern of glioblastoma is recurrence in the wall of the resection cavity (90%) followed by marginal recurrence at <2 cm from the margin of the resection cavity.[38] Therefore, onion skin resection centring on the initial resection cavity might be added at second surgery. In any case, resection of the wall of the initial resection cavity will be performed. Therefore, both the posterior portion and the medial wall of the SMA proper are very likely to be resected. The risk of white matter injury to the primary motor fibres, such as the corticospinal tract, also may be higher than at initial resection. We emphasise that surgical decision-making for recurrent glioma in the SMA should carefully consider the potential benefits and risks.

The major limitation of this retrospective study is the small number of patients, which limits the statistical power of the results. Large numbers of cases of surgical resection limited to this small area (only SMA proper) are comparatively difficult to collect in a single Japanese institute. This study first proposes the importance of the SMA medial wall in surgical resection based on the post-operative neurological deficits. However, our findings could not provide conclusive evidence based on large-scale analysis. The neurosurgeon may have to make the final surgical judgment based on the characteristics of each individual case, especially the grade of malignancy and progression of the tumour.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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